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EDITORIAL

Give a dog-end* a bad name

"Exposure to tobacco smoke and 'well being and health' are incompatible. Discuss." At first glance, this hypothetical examination question poses no great intellectual challenge. Cause and effect have been established beyond reasonable doubt, by means of well-designed pro- and retrospective epidemiological studies, between active cigarette smoking and a statistically significant increase in risk of bronchial carcinoma and coronary heart disease (CHD). It has also been asserted that active smoking can increase morbidity and mortality from a host of other pathological conditions affecting almost all physiological systems. A quick glance down the index to our selected abstracts list will testify to that statement. Serious questions have, however, recently been raised in this and other Journals on the reliability of some data seemingly establishing some of these causal relationships. In the previous issue Professor Ashford (J. Smoking-Related Dis. 1992; 3; 263–274) pointed to the very real problems in assessing smoking-related mortality from all causes and suggested that, not infrequently, a degree of interpretative licence appears to have been exercised in arriving at expected conclusions.

If data on active smoking are being occasionally called into question, there are even greater problems concerning passive smoking and environmental tobacco smoke (ETS). Cardiopulmonary disease, asthma, atherogenesis, lung cancer, leukaemia, retarded growth in children – in these and many more instances, a case has been made for ETS as a major aetiological factor. But assessing the impact of ETS is an exercise made hazardous by confounding variables lurking around every statistical corner. In the case of CHD, for example, some 300 risk factors have at some time or other been identified – by what means is it possible to unravel these data and point the finger with any degree of confidence at ETS *per se* as a major causative element?

In this issue, Dr Armitage (pp27–36) tackles the question of ETS and CHD, and his analysis of 12 major epidemiological studies leads him to conclude that the relationship between ETS and increased risk of CHD "is not proven." He has some cogent remarks to make on the suitability of meta-analyses in the assessment/evaluation of the effects of ETS and about publication bias. Papers with a statistically significant result supporting the point of interest are more likely to be submitted and accepted for publication than are those covering larger numbers of subjects, but where there is a null finding. Further difficulties are encountered when determining inter- and intra-population quantitative exposure to ETS. Should domestic exposure alone be measured, or continual but varying exposure over the course of time? How do you compare groups from different sized households both in terms of numbers of smokers and non-smokers and in the actual area of containment? Is exposure accurately determined by salivary or urinary cotinine concentrations? These are questions which urgently need to be addressed in a meticulous manner to stem the flow of poorly researched or analysed data which could ultimately prove to be counter-productive to the overall public health task.

In an article in the Viewpoint series in the Lancet (1992, 340; 1208–1209) Dr Petr Skrabanek points out another fascinating statistical paradox arising from anti-smoking campaigning over the years. The number of deaths in the UK allegedly attributable to smoking has risen from 50,000 per annum in 1962 to 150,000 at present. However, the number of smokers has fallen

*Editor's note: For our American cousins, dog-end = butt.

from 75% of men and 50% of women in 1958 to 33% and 30%, respectively, 30 years later. What, one is tempted to enquire, is going on? Could it be that smoking is becoming an increasingly convenient scapegoat? In an illuminating couple of sentences, Skrabanek puts it into perspective: "Statements such as 'a half-billion of the world population will be killed by tobacco' are intended to horrify. They tell us nothing about what to advise a 65-year-old widow with rheumatoid arthritis, who smokes 15 a day – *and such information is nowhere to be found.*" (My italics.) Subsequent correspondence (Lancet 1993; 341; 58–59) underscores the importance of resolving such clearly emotional questions.

The Journal of Smoking-Related Disorders is firmly behind all efforts to prevent the young from starting to smoke and convincing older people to stop. But we can also see the dangers inherent in overkill and the use of unsubstantiated generalisations. Campaigners are by nature evangelical in their approach – but the scientific argument has to be built on more solid foundations. To this end, the Editors of the JS-RD together with the publishers, Gardiner-Caldwell Communications (GCC Ltd), are actively canvassing support for an International Congress which will address some of the issues touched upon above. Anyone who would like further information should contact the Managing Editor directly at GCC Ltd, Macclesfield.

Returning to our examination question – it does not, after all, elicit a clear-cut answer. For many, smoking is a pleasurable experience and to many more probably the only pleasurable one left without which their particular 'well being and health' might well suffer deleteriously. That is a fact which certainly the General Practitioner has to take on board when dealing with the individual patient (relevant to the quotation from the Lancet article above). It does and should not in any manner detract from the main thrust of the pathophysiological anti-smoking arguments which will be pursued with vigour in the pages of this Journal.

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